Detection, Recognition and Management of Armillaria and Phellinus Root Diseases in the Southern Interior of British Columbia

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Preface

The root diseases caused by *Armillaria ostoyae* and *Phellinus weirii* reduce timber productivity and affect other resource values over a substantial area of the southern interior of British Columbia. This guide describes how to recognize and detect the diseases, the biology of the fungi, and management options for diseased sites. Information on the fungi comes from published work and the authors' observations and unpublished research. The management options presented are based on results from control trials, especially the Skimikin.

Our intention is to provide forest managers with the information they need to make decisions about disease management options, and not to prescribe set guidelines for treating diseased sites. The guide updates the information published in "*Phellinus weirii* Root Rot: Detection and Management Proposals in Douglas-fir Stands" (Wallis 1976) and "Armillaria Root Disease: A Guide to Disease Diagnosis, Development and Management in British Columbia" (Morrison 1981). It was written for use in the southern Interior.

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introduction

Several root diseases occur in the forests of the southern Interior of British Columbia. Root diseases reduce timber production by slowing tree growth, predisposing trees to windthrow and bark beetles, and causing mortality. The timber volume lost in British Columbia through reduced growth and mortality caused by root disease is estimated to exceed 3.8 million m^3 annually (A. Van Sickle, Pacific Forestry Centre, pers. comm.). Two root diseases, those caused by *Armillaria ostoyae* (Romagn.) Herink and *Phellinus weirii* (Murr.) Gilbn., are widespread in several biogeoclimatic zones.

Root disease fungi are normal components of many ecosystems. Before the widespread exploitation of forests by man, forest stands lasted from one destructive event—abiotic (fire) or biotic (insect or disease)—to the next. Today, in undisturbed seral and climax forest cover types, signs of root disease fungi are not difficult to find. Commonly, 10–20% of trees in seral stands are infected or have been killed. During a period of several hundred years, the incidence of root disease and the inoculum potential of the fungus decline until, in climax stands, often of disease-tolerant species, the root disease fungus and its hosts are in equilibrium. The fungus does not kill the host and the tree cannot eliminate the fungus.

The equilibrium between host and fungus is easily disturbed by harvesting, by establishment of susceptible species, or by management practices. Such disturbance usually increases the incidence and intensity of the root disease by providing stumps which become new food sources for the fungi. During successive 60- to 80-year rotations of susceptible species, there is no opportunity for inoculum to decline. Indeed, evidence suggests that, at a given age, the proportion of stand area occupied by *P. weirii* increases from one rotation to the next (Tkacz and Hansen 1982).

It is therefore important to recognize and identify these root diseases so that appropriate management prescriptions can be made. Failure to do so can result in declining timber productivity, fewer silvicultural options for sites, greater timber supply falldown, and detrimental effects on other resource values in future rotations.

Phellinus weirii

the fungi and their geographical distribution

Both of the host-specialized varieties of *Phellinus weirii* occur in the southern Interior. The cedar variety causes butt rot in western redcedar and is occasionally isolated from Douglas-fir. Anecdotal evidence suggests that the cedar variety does not attack trees of seral species following harvest of old-growth redcedar-hemlock stands and their conversion to seral species.

The Douglas-fir variety attacks and frequently kills conifers other than western redcedar. In the southern Interior, the Douglas-fir variety of *P. weirii* is found west of the Purcell Mountains, except for one record from Radium on Douglas-fir (Fig. 1). The northern record of this variety is a few kilometres north of Williams Lake. The geographical ranges of the two varieties overlap, and it is common for both varieties to occur in subzones of the IDF and ICH which contain both tree species. Hardwoods are immune to both varieties.

The two *P. weirii* varieties are sufficiently host-specialized that they may be distinguished according to their host.

In the southern Interior, the Douglas-fir variety of *P. weirii* occurs in the IDF, ICH, MS and SBS biogeoclimatic zones. Lloyd *et al.* (1990) and Utzig *et al.* (1986) describe these zones for the Kamloops and Nelson Forest Regions, respectively.

This report discusses only the Douglas-fir variety of P. *weirii*.

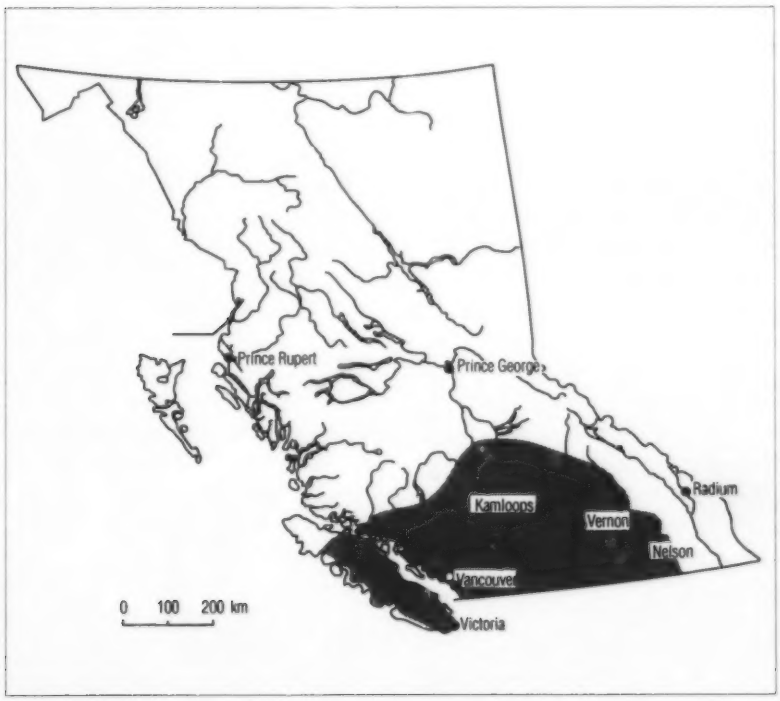

Figure 1.
Distribution of *Phellinus weirii* (Douglas-fir variety) in British Columbia.

Armillaria ostoyae

Two species of *Armillaria* occur throughout the southern Interior. *Armillaria sinapina* Bérubé et Dessureault is found on living and dead hardwoods that were stressed or are overmature, and on stumps of hardwoods and conifers that were colonized after felling. Tests have shown that *A. sinapina* is weakly pathogenic, and field observations suggest that this species is incapable of attacking healthy conifers.

The other species is *A. ostoyae*. Its principal hosts are living conifers, although living hardwoods and shrubs are also attacked. The northern limit of its distribution appears to be a line between McBride, Williams Lake and Bella Coola (Fig. 2). This fungus, which is highly pathogenic, can kill vigorously growing trees throughout a rotation.

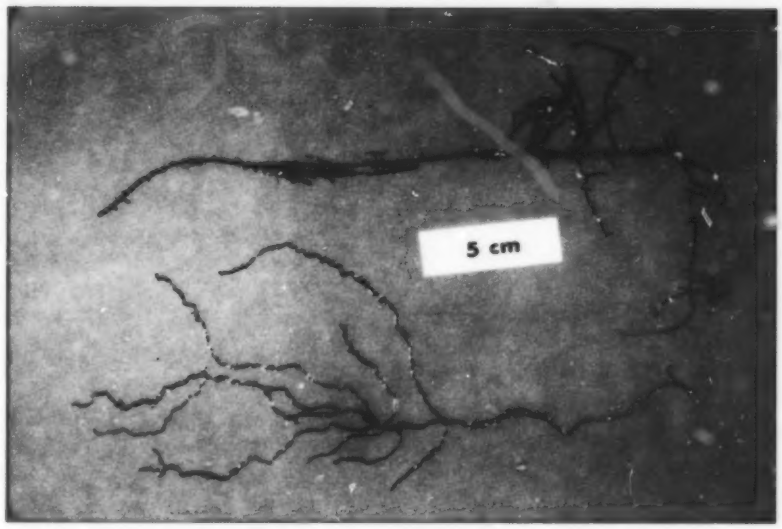

Figure 3. Rhizomorphs of *A. ostoyae* (bottom) and *A. sinapina*.

In the field, *A. ostoyae* and *A. sinapina* are not easily distinguished. Rhizomorphs of the two species differ in amount and branching pattern: *A. sinapina* is a prolific producer of monopodially branched rhizomorphs; *A. ostoyae* produces a much smaller amount of dichotomously branched rhizomorphs (Fig. 3). If Armillaria root disease is attacking living conifers that have not been predisposed by another factor, and if the trees are responding to infection by producing resin at the root collar, then the cause must be *A. ostoyae*. Similarly, recently cut conifer stumps with mycelial fans in the cambial zone and resin at the root collar will contain *A. ostoyae*. The root system of a tree killed by another agent, however, could also be colonized by *A. sinapina*. Although both *Armillaria* species are often present on a site, only *A. ostoyae* will affect management of the site for timber production.

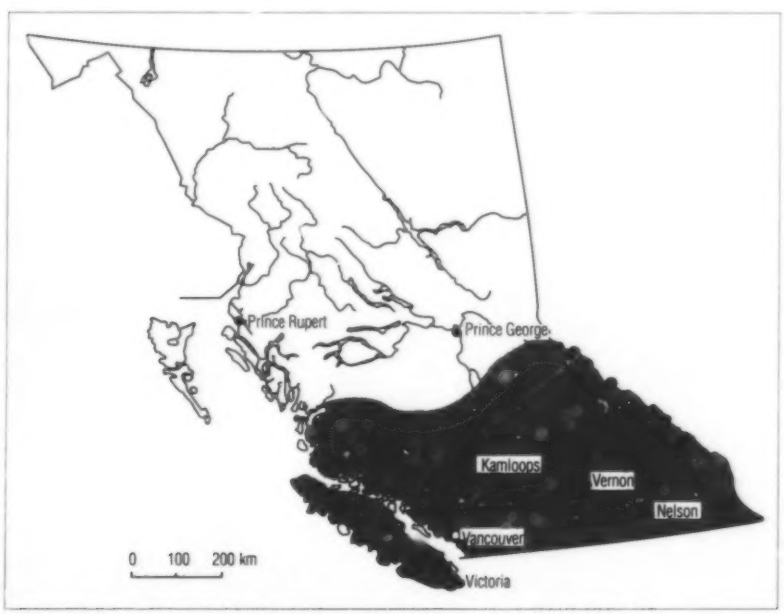

Figure 2. Distribution of *Armillaria ostoyae* in British Columbia.

In the southern Interior, *A. ostoyae* occurs in the IDF, ICH, MS, ESSF and SBS biogeoclimatic zones.

This report discusses only *A. ostoyae*.

fungal biology and disease development

The Disease Cycle

Phellinus weirii and *Armillaria ostoyae* belong to a group of fungi called pathogenic root inhabitants (Garrett 1960). Although behaviour of members of the group differs in detail, they have many characteristics in common.

The life cycle of these fungi consists of two phases: a parasitic phase during which the host is killed, and a saprophytic phase after host death during which the root system is used as a food base. The fungus in the food base is called inoculum. Inoculum potential is defined as the fungus' energy of growth that is available to infect a new host (Garrett 1960). The inoculum potential and longevity of these fungi in a food base depend on the food base size, degree of colonization by the root disease fungus, time since colonization, and activity of insects and other fungi.

To persist on a site, root disease fungi require a continual supply of new food bases. Since mycelium of these fungi is incapable of growth through soil, either contacts or grafts between the food base and roots of potential hosts are essential for spread.

Mycelium of *P. weirii* spreads from a food base over the surface of a host root. Penetration of the root and killing of the host tissue occurs several centimetres behind the mycelial front. The mycelium then spreads to the root collar and onto the remaining primary roots. The tree dies when all primary roots have been killed or when enough roots have been killed to predispose the tree to bark beetle attack or windthrow.

Infection of conifers by *A. ostoyae* at root contacts occurs in a similar manner. Initially, mycelial fans of *A. ostoyae* grow in the outer bark of the host root. As the area of bark colonized increases, mycelial fans penetrate to the cambium. Mycelial fans grow in the bark and cambium to the root collar, then girdle the stem, killing the tree.

A major difference between *P. weirii* and *A. ostoyae* is the production of rhizomorphs by *A. ostoyae*. Rhizomorphs, root-like structures initiated on the food base, grow through soil. When it encounters a host root, the rhizomorph attaches itself to the root and produces lateral branches. These attempt to penetrate the root, thus infecting it. Due to the limited inoculum potential of rhizomorphs, progressive infections are usually initiated only on the roots of trees less than 10 years old.

Another apparent difference is that *A. ostoyae* will rapidly colonize a diseased root system after the tree is killed by the fungus, is cut, or is killed by bark beetles. In contrast, *P. weirii* appears to have limited ability to spread in a moribund root system. Consequently, the inoculum potential and the area containing inoculum could be greater for *A. ostoyae*.

Phellinus weirii appears to spread more rapidly on a living tree's root system than does *A. ostoyae*, because *P. weirii* grows over the root surface rather than in the outer bark and cambium.

The time from infection to death of a tree depends on several factors, including inoculum potential of the fungus and tree species and age. Once infected by either fungus, trees less than about 15 years old are killed within a few years. Time to death increases with age so that *P. weirii* may need 15–20 years and *A. ostoyae* 20–30 years to kill 80- to 100-year-old Douglas-fir. However, not all infected trees are killed. Some of them are able to check the spread of both fungi by producing callus and adventitious roots.

Once colonized, a stump and root system are used as a food source by *A. ostoyae* and *P. weirii*. Undisturbed, the colonized

stump is a secure niche for the fungi which readily form zone lines (the dark lines in decayed wood) as protective barriers to unfavourable conditions or other wood decay fungi. Gradually, over several to many years depending on its size, the food base becomes exhausted. Insects and saprophytic fungi begin to invade through the stump surface and small roots. Eventually, *A. ostoyae* and *P. weirii* are found only in central tissues of large roots and the body of the stump. When *A. ostoyae* and *P. weirii* are no longer present at the surface of the host root, the inoculum is probably not infective.

Disease Dynamics in Managed Forests

In undisturbed forests in which *P. weirii* or *A. ostoyae* is present, diseased trees occur in groups (centres) or as scattered individuals around sources of inoculum. The fungi may be actively attacking trees or quiescent in lesions on tree roots. Disease dynamics in managed forests vary according to the treatment applied to the site.

Following clearcutting

Following clearcutting, stumps of trees colonized by *A. ostoyae* produce a flush of rhizomorphs. Until there is contact between *A. ostoyae* rhizomorphs or diseased stump roots (both fungi) and the woody roots of susceptible vegetation, both root diseases are quiescent. Rhizomorph-initiated *A. ostoyae* mortality begins to appear 4 or 5 years after planting. Mortality initiated by root contact with either fungus appears later (10-15 yr) because roots must grow to the inoculum. In stands older than about 20 years, additional trees are infected after contacting diseased tree roots rather than the inoculum in the stump's root system (Wallis and Reynolds 1965; D. Morrison, Forestry Canada, unpubl. data).

Development of an active disease centre depends on the fungus having adequate inoculum potential and on adjacent trees having roots in contact. Once there is root closure in a stand—that is, root contact between adjacent trees—the diseases have the potential to spread until a physical or non-host barrier is encountered.

Following partial harvest

The root systems of *A. ostoyae*-infected trees which are harvested will be colonized. If the stand was well stocked, the roots of adjacent trees will be in contact and adjacent healthy trees will soon be exposed to inoculum. Moreover, because the food base is newly colonized, inoculum potential will be at its maximum. Repeated partial harvest every 15–20 years often results in severely infested sites on which few trees reach merchantable size. The effect of partial harvest on the disease dynamics of *P. weirii* is not known. Some partially cut Douglas-fir stands, however, are seriously affected by Phellinus root disease.

The effects on disease dynamics of harvesting systems that remove a large proportion of trees, such as shelterwood and seed trees, are probably intermediate between those effects that result from clearcutting and single tree selection.

Following precommercial thinning

As occurs with partial harvest, cutting of infected trees during precommercial thinning can result in rapid colonization of the stump and roots by *A. ostoyae*. The risk of infection to adjacent residual trees depends on the number of root contacts, which in turn depends on distance between trees and other factors. In well-stocked stands, precommercial thinning could increase the number of trees exposed to inoculum and, with the increase in inoculum potential, accelerate the rate of spread of *A. ostoyae* through the stand. The literature on the effects of precommercial thinning on crop tree mortality is contradictory. One study from the northwestern United States recorded a threefold increase in crop tree mortality after precommercial thinning (Koenigs 1969); another recorded no effect (Filip *et al.* 1989). Anecdotal evidence from the southern Interior of British Columbia suggests that thinning does increase crop tree mortality in diseased stands. How thinning affects the disease dynamics of *P. weirii* is not known.

Following brush control

Many broadleaved trees and shrubs, while not primary hosts of *A. ostoyae*, are readily infected and often killed. Frequently the roots of these secondary hosts serve as bridges between conifers. Mechanical or chemical control of brush or broadleaved trees may increase disease incidence in crop trees by increasing inoculum potential and the probability of contact with inoculum. Serious outbreaks of Armillaria root disease in conifers have been recorded following 2,4-D application to broadleaved species in mixed stands with endemic root disease (Pronos and Patton 1978). Treatment of broadleaved trees and shrubs will not affect inoculum levels of *P. weirii* because these plants are immune.

recognizing the diseases

Armillaria and Phellinus root diseases are recognized by the symptoms they induce in their hosts and by signs of the fungi on the host. Conifers attacked by root diseases show an array of symptoms which may be categorized, in approximate progression, as follows: reduction of shoot growth, changes in foliage characteristics, stress-induced reproduction, basal stem indicators, and death. In general, the nature of the symptoms and their rate of development are related to the position of attack and the rate of destruction of the host root system. If the disease progresses rapidly or the host is small, some symptoms may not be evident.

Broadleaved trees and shrubs that are attacked by *A. ostoyae* show reduced growth and sparse foliage before death, and red to brown foliage and mycelial fans at the base of the stem soon after death. Symptoms on these hosts are not as obvious as those on conifers. However, symptomatic conifers are usually found near symptomatic broadleaved trees and shrubs.

Reduction of Shoot Growth

On conifer seedlings and trees up to about 10 years old, *A. ostoyae* and *P. weirii* rarely reduce shoot growth for longer than a year or two because trees are killed within a few years after infection (Fig. 4). By contrast, the slower progress of the disease in older conifers results in a decline in shoot growth which

Figure 4. Growth reduction, chlorotic foliage and stress-induced cones on a 10-year-old Douglas-fir. Note that terminal growth on the dead tree was reduced for only two years.

may be evident for many years. On older trees the shape of the upper crown changes from conical to rounded (Fig. 5) and often the top bends to one side.

Figure 5. Crown symptoms on 75-year-old Douglas-firs. Note the thinning foliage on the tree on the right and reduced growth and rounded crowns on the second and fourth trees from the right, compared to the healthy tree third from the right.

Changes in Foliage Characteristics

On conifers that are killed quickly, foliage turns red or brown as it dries. When the disease progresses slowly, as in older trees, foliage gradually becomes stunted, chlorotic and sparse (Figs. 4 and 5). Usually these changes occur throughout the crown. The 5–7 years of foliage carried by healthy trees declines to 2–3 years as the infection advances.

Stress-induced Reproduction

In response to advanced infection, usually in the season before death, many conifers produce a crop of cones that are smaller but may be more numerous than normal (Fig. 4).

Windthrow, Snags and Stubs

Decay of structural roots by *P. weirii* predisposes trees to windthrow. On these trees the major roots break at the root collar, creating root balls (Fig. 6). In contrast, *A. ostoyae* causes little decay in roots until after the tree has been killed. Trees killed quickly by either disease may remain standing for several years until decay of the stem by slash-decaying fungi causes the stem to break, leaving a stub (Fig. 7).

Many of the symptoms described above are non-specific; that is, they may be induced by a number of biotic and abiotic factors such as blackstain root disease and flooding. It is therefore necessary to examine the root collar and lower bole of the tree for signs specific to Armillaria or Phellinus root diseases. These signs include basal resinosis and mycelial fans, basal lesions, rhizomorphs, and fruiting

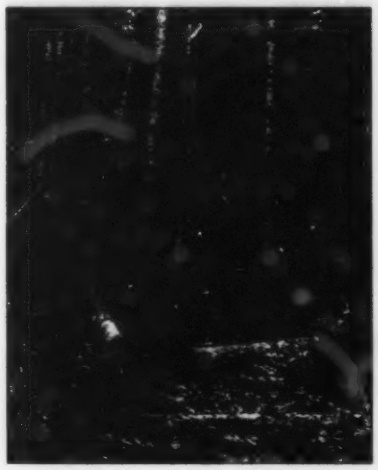

Figure 6. Uprooted Douglas-firs on which roots have broken close to the root collar, creating root balls.

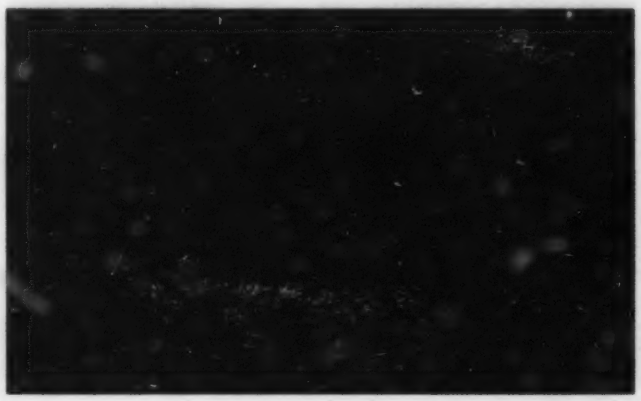

Figure 7. A stub of a diseased tree in an *A. ostoyae* centre.

bodies for *A. ostoyae*; and root surface mycelium and decay for *P. weirii*. Many of these signs are useful for identifying stumps and roots that are within disease centres or on cutover sites, and that are inoculum sources for the next rotation.

Signs of *Armillaria ostoyae*

Conifers in genera that normally have resin canals (*Pseudotsuga, Picea, Larix* and *Pinus*) or that form traumatic resin canals (*Tsuga* and *Abies*) may produce resin that exudes through fissures in the bark of the root collar and lower bole of trees attacked by *A. ostoyae* (Fig. 8). Resin exudation is not usually evident above ground until mycelial fans of the fungus are near or have reached the root collar.

On trees showing basal resinosis, and on those recently killed, creamy white mycelial fans occur in the cambial zone and bark of roots and the lower bole (Figs. 8 and 9). Mycelial fans are the most useful diagnostic characteristic of *A. ostoyae* in woody plants. In trees that have been dead for several years, mycelial fans can usually be found in roots below ground; however, they have disappeared from aboveground parts as a result of competition from other fungi or unfavourable environmental conditions such as desiccation. On conifers, impressions of fans in resin and bark may be present for several years after fans have gone (Fig. 10).

Figure 8. Resin on the bark surface, and white mycelial fans under the bark of the lower bole of a diseased Douglas-fir.

Figure 9. Mycelial fan of *A. ostoyae* growing in the cambial zone of a root.

Figure 10. Impressions of mycelial fans in the inner bark which remain for several years after the fans disappear.

Infections by *A. ostoyae* in Douglas-fir, white pine, western redcedar and other conifers more than 20 years old may be arrested by a strong host response after the fungus has killed cambium above a diseased root (Figs. 11 and 12). Callusing occurs around the margin of the lesion. Recently formed lesions are resinous and have mycelial fans beneath the bark. Later, after the bark sloughs off, the lesions can still be recognized by their shape, location above a root, and the impressions of mycelial fans on the scar face (Molnar and McMinn 1960).

Rhizomorphs of *Armillaria* are initiated on a colonized root system and grow into the soil when a mycelial fan reaches the bark-soil interface. Rhizomorphs in soil and on the surface of roots are usually 1–3 mm in diameter. The rate of growth and distance from the food base they will grow vary greatly among *Armillaria* species. *A. sinapina* forms monopodially branched rhizomorphs which create extensive networks in soil and on root surfaces. *A.ostoyae* forms dichotomously branched rhizomorphs which grow only a few centimetres from the food base (Fig. 3). Rhizomorphs are a useful diagnostic feature when these characteristics are evident.

Mushrooms of *A. ostoyae* occur in clusters on the stem of the host or on soil above diseased roots (Fig. 13). Mushrooms often

Figure 11. Basal lesion caused by *A. ostoyae* on redcedar. Note the callusing at the margin of the lesion.

Figure 12A. *A. ostoyae* lesion on the lower bole of a Douglas-fir. Note the loosened bark and blackened resin.

Figure 12B. Cross-section through the lesion in 12A. Note the active callusing of the lesion.

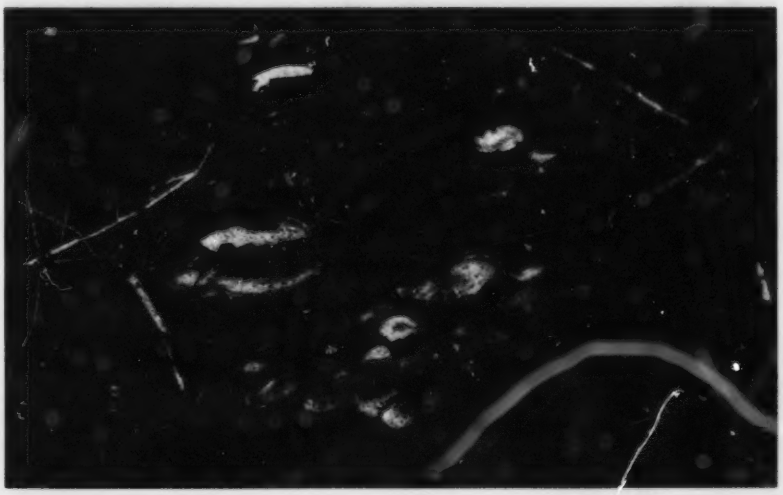

Figure 13. Mushroom fruiting bodies of *A. ostoyae* on Douglas-fir.

occur on or near hosts lacking other signs and symptoms, thereby facilitating surveys of disease incidence. In British Columbia, fruiting occurs from mid-summer to mid-autumn depending on weather. Precipitation and favourable temperatures are required to initiate fruiting and for mushroom development. Mature mushrooms of *A. ostoyae* are 10-20 cm tall, have a cream to brown cap 5-10 cm wide, and have a distinct ring on the stem. Mushrooms that develop slowly as a result of cold or dry weather may have short thickened stems and small thick caps.

In conifers, wood with incipient *A. ostoyae* decay is stained grey to brown, often with a water-soaked appearance. Later, decayed wood becomes yellow-brown and stringy and is finally reduced to a very wet stringy rot with pale yellow flecks (Fig. 14). Decayed wood of broadleaved hosts is water-soaked and white to yellow in colour, becoming spongy and ultimately gelatinous.

Signs of *Phellinus weirii*

On recently dead trees and those showing crown symptoms, root-surface mycelium is the most useful sign for diagnosing *P. weirii* (Fig. 15). The dense, white mycelium is found on diseased roots. Near the soil surface and at the root collar, the surface mycelium may be covered by brown, crust-like mycelium (Fig. 16).

Figure 14. Yellow stringy advanced decay of *A. ostoyae* in a Douglas-fir root.

Figure 16. Dark brown crust and white mycelium of *P. weirii* on the surface of a Douglas-fir root.

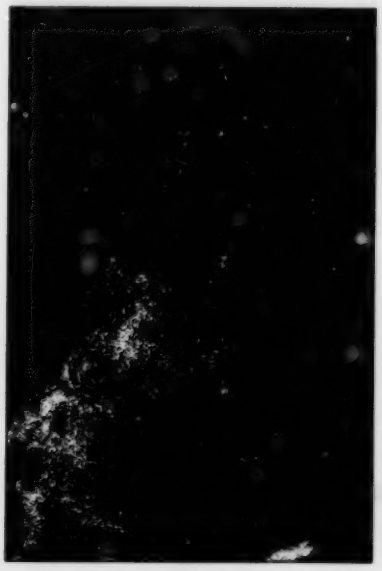

Figure 15. White surface mycelium of *P. weirii* at the root collar of a young Douglas-fir. Note the red-brown crust above the root collar.

Figure 17. Advanced laminated decay of *P. weirii*. Note the small pits in the wood.

The decay of *P. weirii* is its most diagnostic characteristic. The advanced decay is laminated (Fig. 17); that is, the annual rings separate. Brown, hair-like mycelia called setal hyphae are found between the laminations (Fig. 18). Advanced decay occurs in the roots and lower bole of standing dead and windthrown trees. Incipient decay of *P. weirii* is a red-brown stain that is found on the cut surface of stumps and in roots of diseased trees.

Fruiting bodies of *P. weirii* are rare in most years, and hence are of limited value as diagnostic tools. Found on the undersurface of fallen stems, they are light brown with a white margin when young (Fig. 19), becoming dark brown with age.

Figure 18. Brown, hair-like setal hyphae between the layers of wood decayed by *P. weirii*.

Figure 19. Newly formed fruiting body of *P. weirii* on the lower surface of a fallen stem.

disease detection and assessment

Disease Signatures

Armillaria and Phellinus root diseases are detected by their signatures, which are the diagnostic symptoms and signs of the diseases.

On forest cover maps, polygons of age class 3 and older, having significant hardwood components and stands with a lower than expected site class or reduced crown cover, should be suspect for root disease. Similarly, stands that show a mosaic of conifer and hardwood types on aerial photographs may be diseased (Fig. 20).

During ground reconnaissance and surveys, the observer should note symptomatic trees, recently dead trees, snags, stubs and root balls. In plantations, one to several symptomatic or dead young trees will be evident around diseased stumps of the previous stand (Fig. 21). In older stands, stubs, snags, recently dead trees and symptomatic trees can be seen from the center to the margin of the disease patch (Fig. 22). Often an abrupt change in species composition will indicate the margin of a root disease centre—for example, from Douglas-fir to birch or aspen (Figs. 23 and 24), or from mixed seral conifers to western redcedar or redcedar-hemlock (Fig. 25). In each stand, one or more trees at the margin of the patch should be examined for symptoms and signs specific to Phellinus or Armillaria root disease. The observer should examine trees that show advanced crown symptoms, thus ensuring that the critical signs are present. The most useful signs and symptoms are root surface mycelium and laminated decay for *P. weirii*, and basal resinosis and mycelial fans for *A. ostoyae*.

Armillaria ostoyae is known to occur in a high proportion of polygons in the ICH. However, Armillaria root disease may be difficult to detect in overmature redcedar-hemlock stands because, although basal lesions are found occasionally, trees with crown symptoms or basal resinosis are uncommon. When writing pre-harvest prescriptions for redcedar-hemlock stands, foresters should assume that *A. ostoyae* is present on the site.

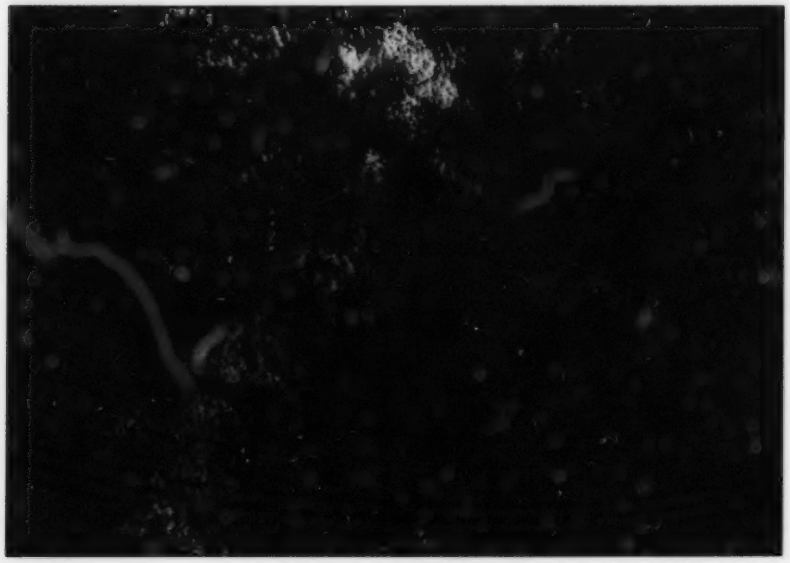

Figure 20. Aerial photograph of Douglas-fir–lodgepole pine stand in which birch and aspen have occupied the root disease centres. (Photographed by Ken Day)

Figure 21. A disease centre around stumps in a Douglas-fir plantation. Note that trees show a range of crown symptoms.

Figure 22. Stubs, snags and recently dead and symptomatic trees seen from the centre towards the margin of a disease patch.

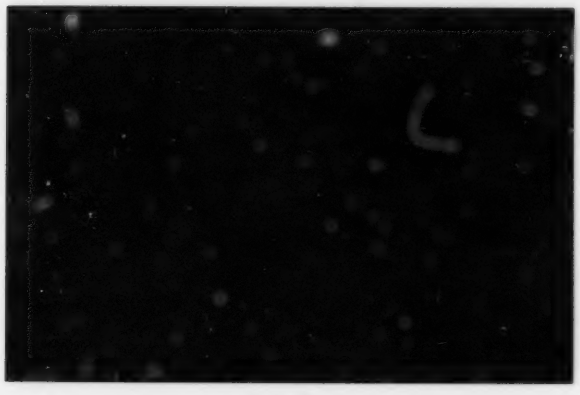

Figure 23. *Armillaria ostoyae* centre in which aspen has replaced Douglas-fir.

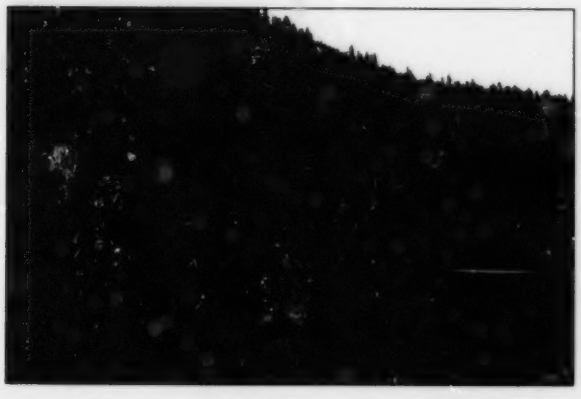

Figure 24. Mosaic of birch and Douglas-fir patches following killing of Douglas-fir by *P. weirii*. Note the scattered red-crowned Douglas-firs.

Figure 25. Succession to redcedar (foreground) following killing of Douglas-fir (background) by *P. weirii*.

Disease Assessment

As a component of the site, root disease must be quantified and qualified at the time of resource planning. Comprehensive disease information will aid foresters with block development in the cutting permit, with the pre-harvest silviculture prescription, and with treatment priorities.

Survey design varies from regularly or randomly spaced transects to systematically spaced variable and fixed radius plots. The ground survey method developed for *P. weirii* (Bloomberg *et al.* 1980a, 1980b), as well as modifications for multiple-disease recording and stratification by infection intensity (Bloomberg 1983), is also applicable to surveys for Armillaria root disease. This transect sampling system uses randomly located sets (grids) of lines to estimate the incidence, distribution, and area of root disease. Estimates of total area of root disease are derived from the length of transect intersecting root disease centres and the probability of occurrence. Random location of gridlines in a stand results in independent estimates for each grid; hence, the variance of their means can be estimated. However, the Bloomberg method is difficult to apply in logged, burnt or open stands with diffuse disease distribution due to difficulty in locating boundaries of infection. For that reason, Kellas *et al.* (1987) used systematically located transects with variable-sized plots around selected stumps to assess infection by *A. luteobubalina* Watling and Kile in regeneration, regrowth and residual trees.

The pixel survey (H. Merler and D. Norris, B.C. Ministry of Forests, unpubl. report) uses a series of systematically located transect lines run perpendicular to a base line. Pixels are plots that may vary in size (3 by 25 m, 10 by 10 m, etc.) according to the age and type of stand being surveyed. They are contiguous on both sides of the transect line. Each pixel is searched for symptoms and signs of root disease and is recorded as diseased or not. Area sampled is determined from pixel width and length of transect line. The interval between transect lines is adjusted to give the desired survey intensity. Combined with stratification of the stand to be surveyed, the pixel survey provides information on the distribution, intensity and identity of root disease.

Survey intensity should be determined by the information needed. For resource planning, sampling within the forest cover strata that make up the resource unit should be thorough but not overly intense. Sampling can be done at between a 1 and 5% area sample and render general disease incidence by stratum type. For cutting permits and block development, the sampling scheme must be able to delineate infected strata for a minimum treatment area - that is, the smallest area that will be treated with proactive disease management techniques. Note that an infected stratum does not refer to infection centres only; treatment of infection centres only is not recommended.

control strategies

The critical points in the disease cycle of *Phellinus weirii* and *Armillaria ostoyae* are: 1) infecting, killing and colonizing a food base; 2) surviving in the food base; and 3) spreading from the food base to a susceptible host. If the fungi fail to accomplish any one of these steps, the disease cycle is broken. Given these critical points, there are several strategies for reducing damage caused by the diseases: 1) crop rotation; 2) inoculum reduction; and 3) increasing the number of disease escapes. It is difficult to stop the spread of any outbreak of root disease once it has started, so measures to prevent or reduce root disease are best applied when the crop is established.

Crop Rotation

Carry-over of root disease from one rotation to the next depends on survival of the fungus in a food base until a susceptible host root contacts inoculum capable of causing infection. Depending on factors such as stump size and degree of colonization, a period of 20–30 years would be required for inoculum of *P. weirii* and *A. ostoyae* to become non-infective (D. Morrison, Forestry Canada, unpubl. data) During this period, a diseased site could be either dedicated to another use, such as range, or regenerated to a non-host tree species. When a non-host species is used, the length of the period would be a rotation. The non-host species would be established where inoculum is localized or on the entire site if inoculum is scattered. It is important that infested parts of a site be kept free from susceptible species.

Inoculum Reduction

The amount and longevity of inoculum in stumps and roots can be reduced by fumigation or mechanical removal. *A. ostoyae* was eradicated from stumps *in situ* by five different fumigants (Filip and Roth 1977) and *P. weirii* by four different fumigants (Thies and Nelson 1982). Although effective, fumigation is expensive to apply and can be hazardous to operators. Application of fungicides to the soil at the root collar of trees exposed to *A. ostoyae* inoculum did not protect the trees from infection (Filip and Roth 1987).

Mechanical removal of colonized stumps and roots is an effective method for reducing losses from root disease in the next rotation. Twenty years after treatment at the Skimikin trial, cumulative mortality in stumped plots was 0.2-3%. In control plots it was 4.7-27% (Morrison *et al.* 1988). Mean annual increments at this site are 5.7 m^3/ha per year in the stumped block compared to 2.9 m^3/ha per year in the untreated block (MacKenzie 1991).

Increasing Disease Escapes

Susceptible trees growing on infested sites but not becoming infected are called escapes. These trees avoid infection because their roots do not contact inoculum. The number of escapes could perhaps be increased if planting spots that avoid inoculum were selected, and if a mixture of susceptible and non-host species were planted. At the Skimikin trial after 20 years (Morrison *et al.* 1988), the number of trees in disease centres was lower in mixed plots of susceptible and immune species than in plots of susceptible species only.

disease management options

The goals for root disease management are to regenerate harvested areas, improve timber production and reduce losses to root disease during the rotation. Inoculum reduction by stumping is the best way to ensure these goals are achieved.

No Treatment Option

Wildlife habitat, topographic and edaphic limitations, visual quality objectives, or other constraining factors may dictate that the no treatment option be used for a diseased site. Disease openings in otherwise uniform landscapes provide aesthetic relief. Diversity in habitat for cavity nesting birds and browse availability for ungulates may be enhanced by the presence of root disease. Browse enhancement may be temporary because many species are hosts for *A. ostoyae*. However, the consequences of choosing the no treatment option cannot be ignored. Where timber production is the principal management objective for the site, the no treatment option could increase spread of the disease, reduce productivity, and shift vegetation to more disease-tolerant tree species.

Free-growing requirements under the pre-harvest silviculture prescription may not be reached. As well, sites generating less timber than forecasted will have effects on timber supply. Timber supply forecasts must be adjusted accordingly.

Treatment Options Following Harvest

Inoculum reduction

Inoculum longevity and infectiousness are greatest in the lower part of the stump and large diameter roots near the stump. Therefore, to be effective, stumping must remove the stump body and as much of the root system as possible. Small diameter roots (<2 cm) and pieces of larger diameter roots may break off during stump removal. The viability of inoculum in these pieces is limited by their small size and by their having been disturbed, broken and exposed to invasion by soil saprophytes. These pieces often have sufficient inoculum

Treatments That Do Not Reduce Root Disease

- Fire as a management tool is effective only in influencing the type of vegetation that returns to the site. This may be important in establishing a mixture of pioneer species. Fire will not reduce the amount of inoculum in stumps and roots.
- Application of chemicals, such as borax, to stump tops does not reduce the amount of *P. weirii* or *A. ostoyae* in stumps because spores are not important in spreading the diseases.
- Fertilization of young, diseased stands may delay symptom expression but will not affect disease spread or amount of mortality.
- Wide spacing of susceptible species is not recommended for controlling root disease unless these species are mixed with resistant or immune species.

potential to kill young regeneration trees which contact them. However, observations in stumped plots suggest that the inoculum piece and the killed tree rarely constitute a long-term threat because the inoculum has lost viability in the time it takes roots of adjacent trees to grow to it.

Use of wide-tracked excavators to push-fall trees or remove stumps after conventional logging is a major advance over a cat with brush blade (Fig. 26). The excavator removes more roots from the soil, does not miss stumps, and minimizes site degradation.

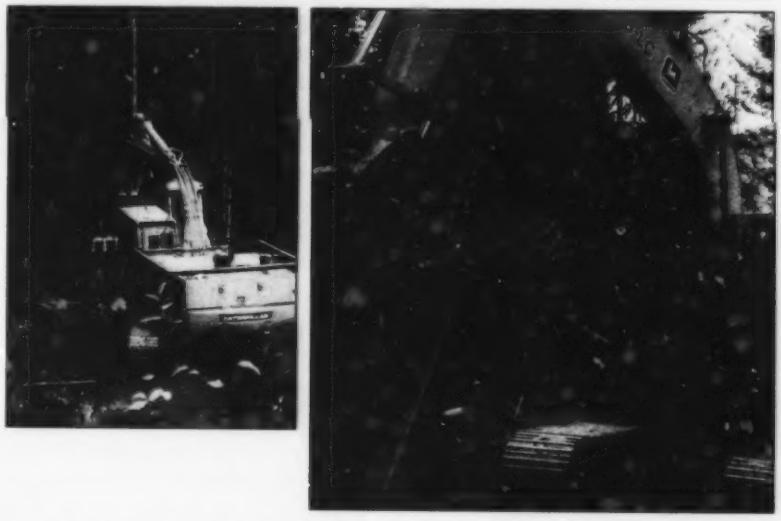

Figure 26. Excavator push-falling a diseased stand (Left) and removing stumps after conventional harvest (Right).

In the absence of soil and topographic constraints, diseased stands - particularly those with *A. ostoyae* - should not be harvested by any selective cutting system. Increased inoculum and inoculum potential following colonization of stumps by the root disease fungi puts residual trees and regeneration at high risk. Instead, mechanical stump removal can be adapted for treating diseased stands by clearcutting, seed tree or single tree/species selection. Clearcut size and shape can be adjusted to meet regeneration and non-timber constraints.

Use of non-host or poor-host tree species

Where soil or topographic constraints preclude inoculum reduction, management for non-host and poor-host species in the next rotation offers the best chance of maintaining timber production. However, the silvics of less susceptible tree species may limit their specific site application.

The choice of non-host or poor-host species is limited for both fungi, but especially *A. ostoyae* (Table 1). The susceptibility ratings in Table 1 are based on inoculations and field observations for *P. weirii* (Wallis 1976;

Table 1. Species susceptibility to *Phellinus weirii* and *Armillaria ostoyae* [a]

Susceptibility	*P. weirii*	*A. ostoyae*
Susceptible	Douglas-fir *Abies* spp.	Douglas-fir *Abies* spp. spruce
Moderately Susceptible	larch spruce hemlock	lodgepole pine hemlock cedar
Tolerant	lodgepole pine white pine ponderosa pine	ponderosa pine ?birch
Resistant	western redcedar	larch
Immune	poplar aspen birch	

[a] There is little difference in susceptibility to *A. ostoyae* among conifers less than 15 years old.

Thies 1984), and on field observations only for *A. ostoyae* (Morrison 1981).

When diseased, conventionally harvested sites are planted, it is not practicable to try to identify stumps that harbour root disease inoculum, or to determine the distribution of inoculum in the stump roots. Consequently, all planting spots on diseased sites must be considered high hazard for root disease, for all but immune species.

For sites infested by A. ostoyae

No tree species shows resistance to *A. ostoyae* before age 15, with the possible exception of birch (H. Merler, B.C. Ministry of Forests, unpubl. data). After that, western larch shows increasing resistance to the fungus until, at about age 25, trees in contact with inoculum are either not infected or infection is checked. Use of larch to regenerate diseased sites is recommended wherever the ecosystem permits. Early survival of larch could perhaps be enhanced if it was planted in a mix with birch.

Use of species from the susceptible and moderately susceptible categories (Table 1) to regenerate diseased sites is not recommended, except in mixtures with birch and perhaps larch. Mortality, particularly juvenile mortality, should be expected whenever susceptible species are regenerated. The amount of mortality will depend on the amount and potential of inoculum and could be high enough to preclude free-growing status.

A very high proportion of plantations in the ICH have some level of *A. ostoyae* damage. Many of these plantations were established after the harvest of decadent redcedar - hemlock forests. Diseased stumps can be identified 3-5 years after harvest when mycelial fans reach the stump and when mushrooms are produced on the stump and roots. These stumps occur in small groups scattered through the cutover. Even on favourable terrain, it is impracticable to remove the very large root systems before planting in order to remove the small proportion of diseased ones. A delay in regenerating the site to allow diseased stumps to be identified would allow other problems to develop, such as the establishment of brush and susceptible tree species. The only practical treatment appears to be to establish species mixtures or poor-host species.

For sites infested by P. weirii

Hardwoods are immune to the Douglas-fir variety of *P. weirii*; redcedar is resistant; and, except when challenged by high inoculum potentials, pines are tolerant. At the Skimikin trial, no lodgepole pines have been killed by *P. weirii*. Hardwoods, redcedar or pines, alone or mixed, are recommended for regenerating diseased sites. When constraining factors dictate, immune, resistant or tolerant species could be mixed with susceptible species, but again mortality among susceptible species must be expected.

For sites infested by both fungi

Results from the Skimikin trial (Morrison *et al.* 1988) indicate that when both diseases occur on a site, a mixture of lodgepole pine (or ponderosa pine, larch and perhaps birch has the best chance of producing a stocked stand.

Remedial Treatments for Diseased Stands

What can be done to reduce losses in immature stands with active root disease?

In young stands, one option is to begin again following an inoculum reduction treatment or to change to a non-host or poor-host species. If disease levels in plantations of susceptible species are high, few of the trees established at the beginning of the rotation will live to harvest. Although the option to begin again may be unattractive due to loss of time and investment, the treatment could double yields. Furthermore, benefit-cost ratios for the begin again and no treatment options are similar (MacKenzie 1991). A second option for raising stocking to acceptable levels is to plant or encourage naturals of tolerant, resistant or immune species (Table 1) in the disease centres.

During precommercial thinning (spacing), poor-host and non-host species can be preferred and used to create barriers to disease spread between trees of susceptible species. Where disease cen-

tres are few and scattered, barriers to the spread of *P. weirii* can be created by felling a strip of healthy trees around the centre.

It is preferable to forego spacing in *Armillaria*-infected stands than to risk increasing inoculum potential by cutting diseased trees, particularly if diseased trees are callusing basal lesions.

Based on a total chance plan of an area, priority may dictate harvest of diseased, merchantable, but not yet mature timber. Where disease centres are discrete, centres could be pushover-logged or stumped following conventional logging. If this is not possible, they could be regenerated to a non-host or poor-host species after conventional harvest. The area harvested must include infected non-symptomatic trees at the margin of the centre. In the case of *A. ostoyae*, two trees beyond a tree with visible disease signs should be removed, and for *P. weirii*, three trees.

When a diseased stand is proposed for partial harvest and constraining factors preclude stumping, current and future losses can be reduced if the stand contains two or more species differing in susceptibility to the root disease. In a Douglas-fir - larch stand with *A. ostoyae*, for example, Douglas-fir can be harvested and larch regenerated under larch seed trees (Fig. 27). Similarly, in a Douglas-fir - lodgepole pine stand with *P. weirii*, Douglas-fir can be cut and pine regenerated. The residual species must be in the tolerant, resistant or immune categories of Table 1.

Where partial cutting has been done on one or more occasions, inoculum may cover most of a site and, as a result, few trees will reach merchantable size. There are no remedial treatments for diseased, partially cut stands that do not include liquidating the stand. On these sites, which often have visual, wildlife or regeneration constraints, 0.5- to 1-ha blocks scattered throughout the stand should be mechanically treated to remove recent and old stumps. To rehabilitate the site, one-third of the blocks could be harvested at intervals of about 10 years.

Figure 27. Larch seed trees following removal of Douglas-fir attacked by *A. ostoyae*.

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